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Gut Microbiota in Postprandial Immunometabolism: A Systematic Review

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ABSTRACT

Gut microbiota is critical for human health, contributing to various biological processes, such as immune responses and metabolism. Postprandial immunometabolism is the metabolic and immune activity that occurs after a meal and is influenced by the gut microbiota. In this review, we discuss how gut microbiota influences postprandial immune responses and metabolism. We explore how short-chain fatty acids (SCFAs) and other microbial metabolites influence immune cell activation and metabolic pathways (nutrient breakdown) during the postprandial period. Imbalances in the gut microbiota (dysbiosis) can cause immune and metabolic dysfunction, which may contribute to metabolic diseases such as obesity and type 2 diabetes. Additionally, nutrition strategies such as probiotics and prebiotics can promote a healthy microbiota, enhancing postprandial metabolic and immune responses. This review discusses the latest research and the potential of microbiota manipulation to maintain metabolic health and prevent metabolic diseases.

Keywords: *Gut Microbiota, Postprandial Immunometabolism, Immune Modulation, Metabolic Health, Dysbiosis, Short-Chain Fatty Acids (SCFAs), Immune System, Postprandial State*

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1. Introduction

This review comprehensively integrates existing knowledge of the gut microbiota's impact on postprandial immunometabolic responses, a key determinant of health and vulnerability to disease. In particular, the complex interactions between the gut microbiota and innate and adaptive immune responses are crucial for intestinal homeostasis and inflammation (Yoo et al., 2020). These complex interactions, involving microbial communities and their products, play a crucial role in regulating systemic energy metabolism and susceptibility to metabolic diseases (Spiljar et al., 2017, p. 1). The microbiota's metabolites regulate the lipid and glucose metabolism pathways, affecting immune responses and gut barrier integrity (Abeltino et al., 2024; Fu et al., 2023, p. 2). This interaction highlights the microbiota's function as an important metabolic organ, affecting nutrient absorption and well-being (Valencia et al., 2025). Disruption of this microbial community (dysbiosis) has been strongly linked to metabolic and immune diseases, underscoring the need to better understand the mechanisms by which gut microbes regulate host immunometabolism (Boulangé et al., 2016, p. 1; Zhou et al., 2024, p. 3).

2. Gut Microbiota Overview

The human gut is home to a diverse and dynamic microbial community of bacteria, archaea, fungi and viruses that affect host health (Anwar et al., 2021). This complex ecosystem, which varies greatly between individuals, is mostly comprised of bacterial species from the phyla Bacteroidetes and Firmicutes, although other phyla such as Proteobacteria, Actinobacteria, Fusobacteria and Verrucomicrobia are also present (Iacomino et al., 2024, p. 1; Krishnamurthy et al., 2023, p. 2). Although the proportions of these microorganisms vary between individuals - and even within individuals -

a healthy microbiota typically displays high diversity, rich microbial gene content, and a stable core microbiota (Hou et al., 2022, p. 136; Matute & Iyavoo, 2023, p. 2). This variability notwithstanding, a core of about 14 genera consistently present in a large number of healthy adults has been identified (Karl et al., 2018, p. 3). The exact composition of the gut microbiota's taxonomic and functional repertoire is under constant refinement as "omics" technologies and improved cultivation methods develop (Eid et al., 2017, p. 2).

3. The Postprandial State: Metabolic View

The postprandial period, accounting for more than 16 hours a day, is a complex phase where ingested nutrients, hormones, and metabolites derived from the diet interact (Meessen et al., 2019). It triggers a series of physiological responses to absorb, metabolise, and store nutrients, under the control of a complex network of hormones, and the influence of the gut microbiota (Holst et al., 2016). This complex interplay prevents hyperglycemia and hyperinsulinemia while ensuring proper nutrient distribution to meet energy needs and store excess nutrients (Dimitriadis et al., 2021, p. 163). For example, the gut microbiota affects the release of different gut hormones, such as GLP-1, GIP, and PYY, essential for insulin sensitivity, glucose tolerance and appetite (Martin et al., 2019, p. 1; Rastelli et al., 2019). For example, short-chain fatty acids generated from the fermentation of dietary fiber by the microbiota induce the secretion of GLP-1, and peptide YY, which play roles in glucose metabolism and satiety (Feng et al., 2023, p. 366; Santos-Marcos et al., 2023, p. 9).

4. Immune-mediated Effect of Gut Microbiota

This review delves into the complex interplay between the gut microbiota and immune responses, with a focus on immune response in postprandial phase and subsequent crosstalk in metabolic regulation (Abeltino et al., 2024). The gut microbiota plays a key role in regulating immune responses and metabolic processes through the production of metabolites such as short-chain fatty acids, which activate immune cells, and strengthen the mucosal barrier (Yusfarani et al., 2025; Zhou et al., 2024, p. 2). These microbial factors (e.g., short-chain fatty acids) not only contribute to maintaining the gut barrier but also exert systemic immunomodulatory effects that influence the development of metabolic diseases (Spiljar et al., 2017, p. 1). In particular, gut microbial anaerobic fermentation of non-digestible carbohydrates produces short-chain fatty acids (acetate, propionate and butyrate) that play a key role in these beneficial effects on host health (Chambers et al., 2018, p. 198). These gut microbiota-derived metabolites, in particular, short-chain fatty acids, play a role in regulating host immunity and metabolism through epigenetic modifications, cytokine production, and interactions with G protein-coupled receptors (GPCRs) (Li et al., 2025, p. 1; Zhang et al., 2019, p. 2).

5. How the Microbiota Modulate Postprandial Immunometabolism

Short-chain fatty acids, key gut metabolites, are short-chain carboxylic acids with fewer than 6 carbon atoms that play important roles in host health and disease (Yao et al., 2020). Acetate, propionate and butyrate are the best-studied SCFAs that are mainly produced by microbial fermentation of undigested carbohydrates in the colon (Kim et al., 2024, p. 2; Rooks & Garrett, 2016, p. 344; Zhang et al., 2023, p. 1). In particular, the phylum Firmicutes are key producers of butyrate, whereas acetate and propionate are mainly produced by Bacteroidetes (Levy et al., 2016, p. 1590). These SCFAs are found at elevated levels in the gut, with peak concentrations in the cecum and the proximal colon, where they play a vital role in regulating various cellular functions such as gene transcription, chemotaxis, differentiation, proliferation and apoptosis (Shibata et al., 2017, p. 3; Zhang et al., 2019, p. 2). Beyond their direct actions on cells, SCFAs make a significant contribution to the gut metabolome, and are essential for the development and function of immune cells (Gonçalves et al., 2018, p. 568). For example, SCFAs regulate immune cell function by binding to G protein-coupled receptors (GPRs), such as GPR43 and GPR109A, on immune cells and modulating their differentiation (Caballero & Pamer, 2015, p. 240).

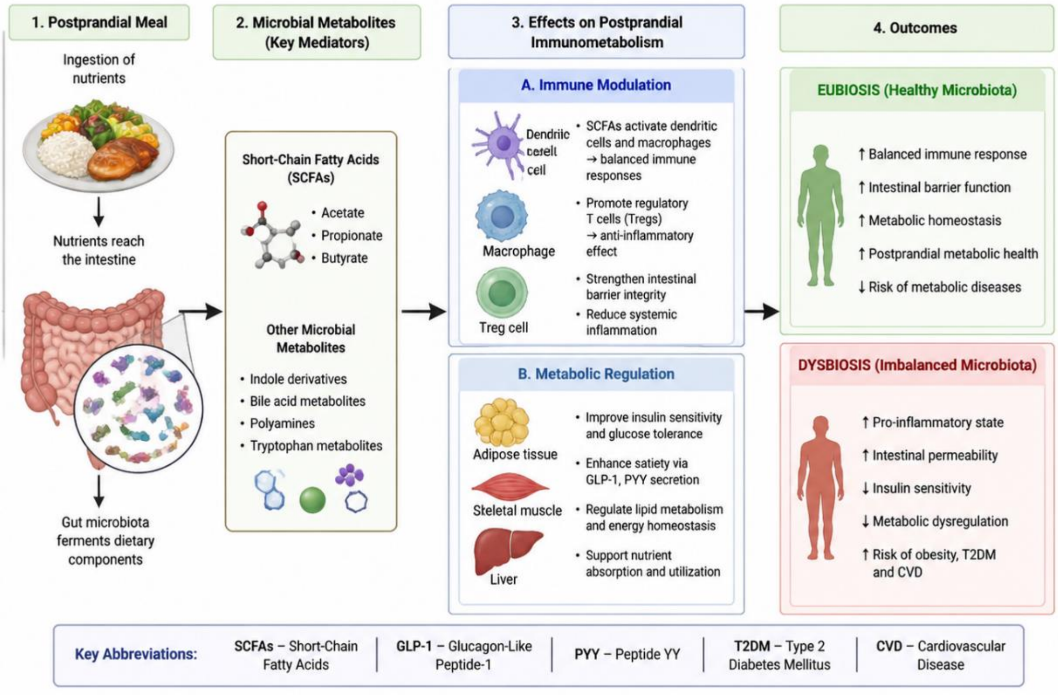

Figure 1. The role of gut microbiota in postprandial immunometabolism.
Source: Adapted from Abeltino et al., 2024; Spiljar et al., 2017; Fu et al., 2023; Valencia et al., 2025.

As illustrated in Figure 1, after eating, the gut microbiota ferment the unabsorbed components of the food and generate metabolites, including short-chain fatty acids (SCFAs). These metabolites are essential in regulating the immune response and metabolic processes in the postprandial period. Eubiosis results in immune homeostasis and metabolic balance, while dysbiosis causes immune dysfunction and contributes to metabolic diseases including obesity, type 2 diabetes and heart disease.

6. Dysbiosis and Immune Dysregulation after a Meal

The complex interactions between a dysbiotic gut microbiota and systemic immune responses play an important role in the development of metabolic diseases, including obesity and type 2 diabetes (Li et al., 2017, p. 2; Scheithauer et al., 2020, p. 5). This chronic systemic inflammation, also known as "metabolic inflammation", is present in metabolic tissues such as adipose and liver and leads to insulin resistance and dysglycemia (Cani et al., 2012; McPhee & Schertzer, 2015). Recent findings indicate the intestine as a key site of immunogenic changes in diet-induced obesity that affect both innate and adaptive immune responses, which, in turn, affect intestinal barrier integrity and systemic inflammation (Khan et al., 2021, p. 1; Winer et al., 2017, p. 33). Gut dysbiosis has been shown to be critically involved in the loss of integrity of the intestinal epithelial barrier, facilitating increased translocation of bacterial products, such as lipopolysaccharide, into the systemic circulation (metabolic endotoxemia) and systemic inflammation (Nakamura & Omaye, 2012, p. 61; Sittipo et al., 2018).

Table 1. Impact of Dysbiosis on Postprandial Immune and Metabolic Functions

Phase	Healthy Gut Microbiota (Eubiosis)	Dysbiosis (Imbalanced Microbiota)	Consequences
Intestinal Barrier Integrity	Intact intestinal barrier, low endotoxin (LPS) translocation	Impaired barrier function, increased intestinal permeability	Increased LPS translocation into circulation
Immune Activation	Controlled immune activation with balanced immune responses	Excessive immune activation due to microbial components (e.g., LPS)	Chronic low-grade inflammation
Metabolic Regulation	Proper nutrient absorption, metabolic homeostasis	Impaired insulin signaling, glucose intolerance, dyslipidemia	Metabolic dysfunction, excess energy storage
Postprandial Effects	Normal metabolic responses (e.g., insulin sensitivity)	Elevated circulating LPS, inflammatory cytokine production	Insulin resistance, fat accumulation (NAFLD)
Disease Risk	Maintains metabolic health and immune homeostasis	Higher risk of metabolic diseases (e.g., obesity, T2DM, CVD)	Increased risk of obesity, type 2 diabetes, cardiovascular disease

Source: Adapted from Cani et al., 2008; Boulangé et al., 2016; Zhou et al., 2024; Abeltino et al., 2024.

7. Postprandial Gut Microbiota: Effects of Diet

In this review, we examine the complex interactions between different dietary patterns, their effects on the gut microbiota structure and activity, and their downstream effects on metabolic health. In particular, diet is the main driver of the gut microbiota composition and function, which in turn plays a key role in metabolic health and disease risk (Muigano et al., 2025). [79] High-energy, high-fat (saturated), and low-fiber diets, typical of the Western world, lead to a dysbiotic gut microbiota that is linked to obesity, metabolic syndrome and heart disease (Perler et al., 2022). In contrast, dietary patterns high in fiber, polyphenols, and prebiotics promote a diverse, healthy gut microbiota and beneficial metabolic effects (Tripathi, 2025). These dietary approaches have shown great promise in shaping the gut microbiome to prevent the development of chronic inflammatory diseases (Randeni et al., 2024) and obesity-related metabolic disorders (Lou et al., 2024, p. 1). In fact, certain dietary patterns, such as those focused on whole foods such as fruits, vegetables, legumes and grains, foster a diverse and beneficial microbiome, supplying essential nutrients for various health-promoting gut bacteria and having anti-inflammatory properties (Shang et al., 2024, p. 4).

8. Therapeutic Potentials and Perspectives

This review examines existing research on the effects of probiotics and prebiotics on the gut microbiota to enhance postprandial immune responses and discusses potential drug interventions targeting the microbiota-immunity axis (Ashaolu, 2020; Liu et al., 2022, p. 634897). It particularly discusses the immunomodulatory effects of probiotics (which improve gut health and regulate inflammation) and the effects of prebiotics on promoting the growth of beneficial gut bacteria (Zhou et al., 2024, p. 1). Probiotics, which are live microorganisms that confer a health benefit on the host when administered in adequate amounts, have various immune-modulatory effects through different mechanisms, including interaction with immune cells through toll-like receptors, and through the production of beneficial metabolites (Agu et al., 2023, p. 12; Al-Habsi et al., 2024). On the other hand, prebiotics are indigestible substances that selectively promote the growth and/or activity of beneficial bacteria, and increase the production of short-chain fatty acids (SCFAs) like butyrate, which have anti-inflammatory effects and support gut barrier function (Kumari et al., 2024; Montalban-Arques et al., 2015, p. 3). These prebiotics (inulin, polydextrose, fructooligosaccharides and galactooligosaccharides) have been demonstrated to have positive effects on immune responses, gut microbiota diversity, and inflammation, by upregulating anti-inflammatory cytokines and downregulating pro-inflammatory cytokines (Châari et al., 2020, p. 15; Cruz et al., 2021, p. 3; Tunc et al., 2025).

9. Conclusion

This review provides an update of recent developments of the complex relationship between the gut microbiota and the host immunometabolic responses in the postprandial period and reviews key mechanistic findings. In particular, we examine the role of the microbiota, its metabolites and bacterial constituents in immune homeostasis and their impact on immune-mediated disease (Rooks & Garrett, 2016, p. 341). Here, we present the main findings, therapeutic potential and concluding remarks on the importance of considering gut microbiota in postprandial immunometabolism. The microbiota plays an important role in mediating host immune responses, in part by producing metabolites, such as short-chain fatty acids, that regulate immune cell function and create an immunosuppressive environment (Man et al., 2020; Zhou et al., 2024, p. 2). These metabolites (e.g. acetate, propionate, butyrate) are products of dietary fiber fermentation, and can have profound effects on local and systemic immune responses (Basso et al., 2024, p. 3). For example, short-chain fatty acids contribute to immune homeostasis of the gut by inducing and maintaining regulatory Foxp3+ T cells, and can alleviate experimental colitis (Hand et al., 2016, p. 833).

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